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Targeted disruption of the c-src proto-oncogene leads to osteopetrosis in mice

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Abstract

To understand the normal, physiological role of the c-*src* proto-oncogene, a null mutation was introduced into the gene by homologous recombination in mouse embryonic stem cells. Two independent targeted clones were used to generate chimeras that transmitted the mutated allele to their offspring. Intercrossing of heterozygotes gave rise to live born homozygotes, but most of these mice died within the first few weeks of birth. Histological and hematological examination of the homozygous mutants did not reveal detectable abnormalities in the brain or platelets, where *src* is most highly expressed. However, these mutants were deficient in bone remodelling, indicating impaired osteoclast function, and

developed osteopetrosis. These results demonstrate that *src* is not required for general cell viability (possibly because of functional overlap with other tyrosine kinases related to *src*) and uncover an essential role for *src* in bone formation.

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